Influence of ruminal and postruminal carbohydrate infusion on visceral organ mass and adipose tissue accretion in growing beef steers

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Influence of ruminal and postruminal carbohydrate infusion on visceral organ mass and adipose tissue accretion in growing beef steers[1]

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ABSTRACT: Forty crossbred beef steers (243 ± 2 kg of BW) with ruminal and abomasal infusion catheters were used to test 2 hypotheses: 1) visceral mass is responsive to energy input and site of carbohydrate (CHO) infusion and 2) rate and site of adipose accretion are dependent on site of CHO infusion and complexity. Treatments included a pelleted, forage-based, basal diet fed at 161 (LI) or 214 (HI) kcal of ME/(kg of $BW^{0.75}$·d), LI plus ruminal (R-SH) or abomasal (A-SH) infusion of a partial starch hydrolysate (SH), and LI plus abomasal infusion of glucose (A-G). The basal diet was fed in 12 equal portions daily at 2-h intervals, with starch and glucose infused over a 22-h period at rates of 12.6 and 14.4 g/(kg of $BW^{0.75}$·d). After 35 d of infusion, steers were slaughtered; and visceral organ and adipose mass, subcutaneous adipose thickness over the 5th and 12th rib, and LM intramuscular fat concentration were determined. Total intake energy (IE) increased (P = 0.0001) with ME intake. Dietary IE was similar between LI and CHO treatments, but total IE increased ($P < 0.001$) with CHO infusion. Greater dietary ME intake and CHO infusion increased or tended ($P \leq 0.09$) to increase final BW and HCW. As a percentage of empty BW, total stomach complex, rumen, omasum, liver, pancreas, and kidney weights were greater ($P \leq 0.05$) for HI vs. LI. Stomach complex, rumen, pancreas, and kidney weights as a percentage of empty BW were greater ($P \leq 0.05$) for R-SH vs. A-SH. Compared with A-SH, A-G increased ($P \leq 0.02$) total and mucosal weights from the 10-cm sections of the ileum. Increases in rumen mass were associated with no change or an increase in rumen total and mucosal DNA concentrations. Greater dietary ME tended ($P = 0.06$) to increase subcutaneous fat thickness at the 5th rib but did not affect alimentary adipose accretion on an empty BW basis. Omental and total alimentary adipose weights were increased ($P \leq 0.04$) by A-G compared with A-SH. Although SH infusion did not alter adiposity, there was a consistent numerical pattern in total alimentary and subcutaneous fat depots with CHO infusion (A-G > A-SH > R-SH). Our findings demonstrate that increasing ruminal CHO supply results in a disproportionate increase in rumen mass, whereas increasing small intestinal CHO supply does not alter gastrointestinal organ mass. Small intestinal energy in the form of glucose resulted in greater adipose accretion, particularly the omental depot.

Key words: adipose, carbohydrate, energy, steer, visceral organ

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INTRODUCTION

Visceral organs have a significant impact on the partitioning of ME between heat energy loss and NE available for peripheral tissue gain (Webster, 1980; Koong et al., 1985). Accordingly, a considerable amount of data has been generated in an effort to define the relationship between ME intake (**MEI**) and visceral organ mass (Johnson et al., 1990). However, because a majority of approaches are confounded by level of dietary alimentation or changes in diet composition, or both, it is difficult to discern the effects of ME from effects of other dietary factors. Seal and Reynolds (1993) demonstrated a positive relationship between MEI and portal-drained viscera (**PDV**) heat production ($R^2 = 0.612$). Those authors noted a bias in which PDV heat production for diets with greater nondigestible fiber components tended to be underpredicted, whereas those for concentrate-fed animals were overpredicted. Subsequently, we have demonstrated that changes in PDV heat production associated with MEI and dietary composition are a func-

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tion of changes in digestive tract organ mass rather than oxidative capacity (Baldwin and McLeod, 2000; McLeod and Baldwin, 2000).

Small intestinal starch digestion increases PDV and peripheral glucose utilization relative to ruminal starch digestion (Harmon et al., 2001). Additionally, indirect calorimetry measures have shown that retained tissue energy is greater for starch digested in the small intestine than that digested ruminally (McLeod et al., 2001). In the same study, carbon-N balance indicated that the increase in tissue energy with small intestinal starch infusion was realized as adipose tissue. These observations suggested that composition of tissue accretion may be altered by site of starch infusion.

Thus, this study was designed to determine 1) the effects of site of carbohydrate digestion on visceral organ mass, and 2) the effects of site of carbohydrate digestion and carbohydrate complexity on adipose accretion in growing steers.

Table 1. Ingredients and chemical composition of the basal diet

Item	
Ingredient, % of DM	
Orchardgrass hay	89.45
Corn gluten meal	5.00
Lignosulfonate-treated soybean meal	5.00
Trace mineral salt[1]	0.50
Vitamin premix[2]	0.05
Composition	
DM, %	97.0
CP, % of DM	19.5
NDF, % of DM	47.3
ADF, % of DM	28.7
GE, Mcal/kg of DM	4.57
ME,[3] Mcal/kg of DM	2.32

[1]Composition (%): NaCl, 99; Zn, 0.35; Fe, 0.34; Mn, 0.20; Cu, 0.03; I, 0.007; Se, 0.009; and Co, 0.005.
[2]Composition (IU/kg of diet DM): vitamins A, 4,400; D, 880; and E, 0.6.
[3]Based on previous direct determination of ME of the same diet (McLeod et al., 2001).

MATERIALS AND METHODS

Steers and Treatments

All animal procedures were approved by the Institutional Animal Care and Use Committee. Forty predominately Angus crossbred steers (214 ± 2.5 kg of BW) were purchased from a single herd in Maryland (n = 20) or obtained from the Beltsville Agriculture Research Center beef herd (n = 20). Because of technical and animal facility constraints, steers were selected and obtained from these sources 4 mo apart to minimize differences in BW at the initiation of the experiment. Before implementation of the experimental protocol, steers were treated for elimination of internal parasites (Ivermectin, Merial, Duluth, GA), vaccinated (Bovi-Shield 4 and Ultrabac 7, Pfizer Animal Health, Exton, PA), and fed a common diet for 30 d. Steers were surgically fitted with abomasal and ruminal infusion catheters constructed of medical-grade Tygon tubing (64-mm i.d. × 80-mm o.d.) fitted with a 1.3-cm medical-grade Tygon cuff (Norton Performance Plastics, Akron, OH) and a Teflon O-ring (3.8-cm diam., Ace Hardware, Nicholasville, KY) positioned 3.8 cm distal to the tip of the catheter. Before surgical placement of the abomasal catheter, steers were deprived of feed and water for 48 and 24 h, respectively.

Steers were initially sedated by i.v. injection of xylazine (0.07 mg/kg of BW) and ketamine hydrochloride (4.4 mg/kg of BW), intubated, and maintained on isoflurane (1 to 2%) in O_2. Abomasal infusion catheters were inserted 10 cm cranial to the pyloric valve and secured using a combined Lembert and a purse string suture pattern. After at least 10 d of postsurgical recovery, a ruminal infusion catheter was inserted into the dorsal, caudal region of the rumen using similar surgical procedures as described for abomasal catheter placement, under light sedation (xylazine, 0.07 mg/kg of BW) and local anesthesia (lidocaine). After each surgical procedure, steers were given i.m. injections of procaine G penicillin (13,000 units/kg of BW) and banamine (1.0 mg/kg of BW) for 3 d. After surgical placement of ruminal catheters, a recovery period of at least 2 wk preceded commencement of the experimental treatments. During this time, rectal temperature was monitored daily as a sign of infection.

Steers were housed in individual tie stalls (1.2 × 1.7 m) in an environmentally controlled barn (ambient temperature, 18°C; relative humidity, 55%) with a 16-h light cycle and continuous access to water. Throughout the experimental period, steers were exercised for 1 h twice weekly using a motorized rotary walker (Farnam Equipment Co. Inc., Omaha, NE) to maintain animal well-being and feed intake and to avoid potential muscle atrophy. In a randomized complete block design, steers were allotted by BW to 8 blocks containing 5 steers each. Within each block, all treatments were represented and the steers were assigned randomly to treatment. Among and within blocks, the initiation of treatments was staggered such that animal slaughters were conducted at predefined intervals to facilitate tissue sample collection while maintaining a constant 35-d treatment period. Treatments included a pelleted basal diet (Table 1) fed at 161 (**LI**) or 214 (**HI**) kcal of ME/(kg of $BW^{0.75}$·d), LI plus ruminal (**R-SH**) or abomasal (**A-SH**) infusion of a partial starch hydrolysate (**SH**), and LI plus abomasal infusion of glucose (**A-G**).

The basal diet, comprised primarily of orchardgrass hay to minimize dietary starch intake, was fed in 12 equal portions daily at 2-h intervals. Metabolizable energy intake of the basal diet was designed to approximate 1.5 and 2.0 times maintenance energy requirements of growing steers. The ME value of the basal diet was determined to be 2.32 Mcal/kg of DM in a previous experiment (McLeod et al., 2001). Additionally, the basal diet was formulated to meet the rumen-digestible

intake protein requirement for steers in the R-SH treatment and the MP requirement for steers (250 kg of BW) gaining 0.84 kg/d (NRC, 1996). Corn SH (20%, wt/wt) was prepared as described by Bauer et al. (1995) and stored (–20°C) until infused. Glucose infusate solution (25%, wt/wt) was prepared and stored using the same procedures as those described for SH to eliminate processing effects. Based on their respective heats of combustion, infusion rates of 12.6 and 14.4 g/(kg of $BW^{0.75}$·d) were used to achieve isoenergetic infusions of SH and glucose, respectively, and to supply 20% of the total MEI. Further, this amount approximates the maximal small intestinal, digestive capacity of SH in growing beef steers (Branco et al., 1999). Stock infusate solutions were diluted to a final weight of 5 kg using tap water and infused over a 22-h period. The total amount of infusate was equalized among treatments and infusion site by infusion of tap water. Steers were adapted to feed intake and carbohydrate infusate incrementally over the initial 6 d of the 35-d treatment period. Feed offered and orts were recorded daily. Steers were weighed at 0900 twice weekly, and the amount of feed offered and carbohydrate infused was adjusted weekly based on the average BW from the preceding week.

The basal diet was sampled daily, composited weekly, and analyzed for DM (AOAC, 1984). Weekly determinations of DM content were used in the adjustment of the amount of feed offered the following week. A portion of each weekly sample was retained and subsequently composited by animal block at the end of the treatment period. Composited dietary samples were ground through a 1-mm screen in a ultra-centrifugal mill (model ZM-1, Brinkmann Instruments Inc., Westbury, NY), and analyzed (Table 1) for DM, N (model CN-2000 carbon/nitrogen analyzer, Leco Corporation, St. Joseph, MI), GE (model 1241 adiabatic calorimeter, Parr Instrument Company, Moline, IL), NDF and ADF (Goering and Van Soest, 1970), and ether extract (AOAC, 1984). The NDF procedure was modified by the addition of heat stable amylase (Sigma Chemical Co., St. Louis, MO) to all samples during analysis. Additionally, each batch of stock carbohydrate infusate was sampled immediately after preparation for determination of DM and GE, as described above.

Visceral Organs and Tissues

Immediately after the 35-d feeding and infusion period, the steers were slaughtered (0900 and 1100) at the institutional abattoir. To maintain integrity of harvested tissues, only 1 to 2 animals were slaughtered per day. Within each animal block, the order of animal slaughters was determined at the onset of the treatment period, such that all treatments and treatment combinations were balanced among single or multiple animal slaughter dates and animal slaughter pairs. Steers were stunned with a captive-bolt gun, exsanguinated, and the viscera were removed and placed into a visceral cart for separation. Liver was immediately separated from the surrounding connective tissue, weighed, and sampled for composition analysis. The pyloric valve and ileocecal junction were identified, and the cranial and caudal ends of the small intestine were ligated before separation to prevent contamination of tissues, particularly adipose, with digesta contents. Stomach compartments (rumen, reticulum, omasum, and abomasum) were separated from connective and omental adipose tissue, digestive contents were emptied, and the compartments were rinsed with warm tap water, blotted, and weighed. The rumen and reticulum were separated by inverting the reticulorumen and cutting along the reticuloruminal fold. Immediately after weighing, the ventral cranial region of the rumen was identified and sampled for rumen total (with adherent musculature) and mucosal (separated from the muscle tissue) composition. Omental adipose tissue was collected, rinsed with warm saline, blotted, and weighed.

Small intestinal tissue was separated from the mesentery, and the mesenteric adipose tissue was collected and weighed. Small intestinal length was determined by looping the intestine across a stationary board that was fitted with plastic pegs at 1-m increments, without tension to minimize stretching. Intestinal sections (1 m) from the duodenum (1 m caudal to the pyloric sphincter), jejunum (midpoint of the small intestine), and ileum (1 m cranial to the ileocecal junction) were rinsed with physiological saline (0.15 *M* NaCl), weighed, and processed for compositional analysis of total and mucosal tissue. Intestinal mucosa was harvested by taking a 10-cm section from each of the 1-m intestinal (colon, duodenum, jejunum, and ileum) sections, splitting it along the longitudinal axis, and scraping it with a glass slide. The large intestine and the remaining portions of the small intestine were emptied of digestive contents, rinsed with warm physiological saline, blotted, and weighed. Immediately after tissue weights were determined, rumen total and mucosa, liver, colon total and mucosa, duodenal total and mucosa, jejunal total and mucosa, and ileal total and mucosa were partitioned for RNA and DNA (0.3 to 0.5 g frozen in 2 mL of ice-cold extraction buffer, 10 m*M* Tris with 5 m*M* EDTA, pH 8.0, with dry ice), DM (100°C for 48 h), and N (Leco carbon/nitrogen analyzer). All tissues were weighed, processed, and frozen within 45 min of exsanguination.

DNA and RNA Analysis

Frozen tissues were further diluted 1:14 (sample:buffer, wt/vol) in ice-cold extraction buffer (10 m*M* Tris with 5 m*M* EDTA, pH 8.0) and immediately homogenized (model PT10/35, Brinkmann Instruments) on ice by applying three 1-min bursts over 5 min. A 1.0-mL aliquot of each tissue homogenate was placed into a 1.5-mL polypropylene, microcentrifuge tube and frozen (–70°C) for subsequent DNA analysis using the Hoechst 33258 dye-binding procedure of Labarca and Paigen (1980), modified for a 96-well microtiter plate assay using a fluorometer (FL600, BioTek Instruments Inc.,

Highland Park, Winooski, VT). A second 1.0-mL aliquot was placed into a 2.0-mL microcentrifuge tube and assayed for total RNA (Schmidt and Thannhauser, 1945).

Carcass Measurements

Hot carcass weight was recorded immediately after slaughter, and yield grade factors were determined by a qualified meat scientist on 24-h postmortem, chilled (4°C) carcasses according to USDA standards (USDA, 1997). Yield grade factors considered were LM area, subcutaneous fat thickness over the 12th rib, and KPH. Because of the physiological age of the steers at slaughter and the fact that subcutaneous fat deposition occurs in a cranial to caudal manner (Altmann and Pliquett, 2006), an additional measure of subcutaneous fat thickness was determined over the 5th rib. For determination of intramuscular fat, a LM sample from the 13th-rib location was removed from the right side of each carcass and trimmed of visible fat, bone, and connective tissue. The lean portion of the LM was ground and analyzed for moisture and extractable lipid (AOAC, 1984).

Statistical Analyses

All statistical analyses were conducted using the MIXED procedures (SAS Inst. Inc., Cary, NC). Data were analyzed as a randomized complete block design, with steer group as the block (Steel and Torrie, 1980). The model included fixed effects of group and treatment. Single degree of freedom contrasts were used to test the effects of MEI (LI-H_2O vs. HI-H_2O), starch infusion (LI-H_2O vs. R-SH and A-SH), site of starch infusion (R-SH vs. A-SH), and form of abomasally infused carbohydrate (A-SH vs. A-G). One steer subjected to the A-G treatment was removed from the data set due to inadequate intake of the basal diet (60% of feed offered). Additionally, data from 2 steers assigned to the LI-H_2O were eliminated from all statistical analyses because visceral and carcass adipose tissue measures were not consistent with their treatment cohorts (i.e., greater than 2 SD from the treatment mean), exhibiting a phenotype of more mature steers at slaughter. Additionally, the unbalanced number of observations (n = 6, HI-H_2O) for mass of mesenteric fat reflects unrecorded data and the inability to quantitatively recover the tissue. All data are presented as least squares means ± SEM, with the SEM calculated using the least number of observations for each measured variable. Treatment effects were considered significant at $P \leq 0.05$ and as a tendency at $P \leq 0.10$.

RESULTS

Initial BW was similar among treatments (Table 2). Consistent with greater ($P = 0.0001$) amounts of total intake energy (diet plus infusate), empty BW (**EBW**; $P \leq 0.03$) was greater, and final live BW was greater or tended to be greater, for steers fed the basal diet at the high MEI ($P = 0.03$) or infused with starch ($P = 0.08$) compared with those on the LI-H_2O treatment. Site of SH infusion (R-SH vs. A-SH) did not affect dietary or total intake energy. Glucose infusion resulted in a slight decrease ($P = 0.10$) in feed intake energy compared with the A-SH treatment, but total intake energy did not differ between the 2 treatment groups.

Digestive tract (stomach complex plus intestines; Table 3) weight, on a wet tissue basis, increased with greater dietary MEI ($P = 0.001$; HI-H_2O vs. LI-H_2O) and with SH infusion ($P = 0.02$; R-,A-SH vs. LI-H_2O), but was unaffected by site of SH infusion and form of abomasally infused carbodydrate (A-SH vs. A-G). Greater dietary MEI increased rumen ($P = 0.006$), omasum ($P = 0.001$), liver ($P = 0.003$), pancreas ($P = 0.003$), heart plus lung ($P = 0.01$), kidney ($P = 0.001$), and spleen ($P = 0.02$) weights and tended to increase reticulum ($P = 0.06$), large intestine ($P = 0.06$), and total alimentary ($P = 0.09$) and omental fat ($P = 0.09$) weights. Dietary MEI did not affect abomasum, small intestine, and mesenteric fat weights. Rumen ($P = 0.03$), liver ($P = 0.02$), pancreas ($P = 0.01$), and spleen ($P = 0.01$) weights were greater and large intestine ($P = 0.10$) and heart plus lung ($P = 0.06$) weights tended to be greater with SH infusion compared with LI-H_2O. In contrast, omasum, abomasum, small intestine, kidney, and omental and mesenteric fat weights were unaffected by SH infusion. Rumen ($P = 0.02$) and pancreas ($P = 0.03$) weights were greater and spleen weight tended to be less ($P = 0.07$) for ruminal vs. abomasal SH infusion. Site of SH infusion did not affect omasum, abomasum, small intestine, large intestine, liver, heart and lung, kidney, and alimentary fat weights. Visceral organ and mesenteric fat weights were unresponsive to form of abomasally infused carbohydrate, with the exception of a trend for greater large intestine ($P = 0.08$) and spleen ($P = 0.07$) weights for A-SH vs. A-G. In contrast, A-G increased ($P = 0.03$) omental fat weight and tended to increase ($P = 0.06$) total alimentary fat weight compared with A-SH.

Expressed as a percentage of EBW, digestive tract ($P = 0.02$), rumen ($P = 0.03$), omasum ($P = 0.007$), liver ($P = 0.03$), pancreas ($P = 0.02$), and kidney ($P = 0.05$) weight increased with greater dietary MEI (Table 4). Conversely, greater dietary MEI did not affect reticulum, abomasum, small and large intestine, heart plus lung, spleen, and alimentary fat weights as a percentage of EBW. Overall, with the exception of a trend for greater rumen ($P = 0.10$), pancreas ($P = 0.06$), and spleen ($P = 0.07$) weights relative to EBW, SH infusion did not affect visceral organ or alimentary fat weights as a proportion of EBW. However, effect of SH infusion on visceral organ weights as a percentage of EBW was dependent upon site of infusion. Relative to EBW, R-SH increased ($P = 0.008$) stomach complex weight and tended to increase ($P = 0.09$) digestive tract weight compared with A-SH. These increases were a function of greater rumen ($P = 0.005$) and reticulum ($P = 0.07$)

Table 2. Body weight and energy intake for steers fed at low intake (LI) or high intake (HI) and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM[4]	HI-H_2O	R-, A-SH	A-SH	A-G
Initial BW, kg	241.3	243.3	248.4	240.5	240.5	4.7	0.89	0.41	0.36	0.18
Final BW, kg	274.3	289.3	288.0	282.2	294.9	6.9	0.03	0.08	0.88	0.50
Final empty BW, kg	215.7	224.8	232.4	230.5	230.9	4.8	0.02	0.03	0.20	0.76
Total intake energy[5]	313.5	365.0	364.4	360.1	417.8	3.3	0.0001	0.0001	0.88	0.30
Feed energy[5]	313.5	314.0	313.5	306.5	417.8	3.3	0.0001	0.95	0.89	0.10
Infused energy[5]	0.0	51.0	50.9	53.6	0.0	0.03	—	—	—	—

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of a larger *F*-statistic.
[4]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[5]kcal of GE/(kg of $BW^{0.75}$·d).

percentages concomitant with no difference in the percentage of other digestive tract components for R-SH vs. A-SH. Additionally, a larger percentage of EBW was composed of pancreas ($P = 0.004$) and kidney ($P = 0.05$) for R-SH vs. A-SH, whereas the percentage of other service organs and alimentary fat was similar between infusion sites. Large intestine weight as a proportion of EBW tended to be greater ($P = 0.08$) for A-SH vs. A-G, but all other visceral organs were unaffected by form of carbohydrate infused. In contrast, a greater ($P = 0.02$) proportion of EBW was composed of omental fat, and thus alimentary fat ($P = 0.04$), for A-G compared with A-SH, with no observed effect of form of carbohydrate on mesenteric fat.

Length and mass per unit of length of the small intestine (Table 5) were unaffected by dietary MEI, starch infusion, or form of abomasally infused carbohydrate. However, there was an inverse relationship observed between intestinal length and mass per unit of tissue for R-SH vs. A-SH in that intestinal length was greater ($P = 0.03$) and mass per unit of tissue was less ($P = 0.002$) for R-SH compared with A-SH. Section (10 cm)

Table 3. Visceral organ and alimentary tract fat mass for steers fed at low intake (LI) or high intake (HI) and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM	HI-H_2O	R-, A-SH	A-SH	A-G
						Mass,[4] kg				
Digestive tract	12.44	14.07	13.58	12.56	14.83	0.50[5]	0.001	0.02	0.42	0.11
Stomach complex	7.50	8.77	7.83	7.41	9.29	0.37[5]	0.001	0.08	0.04	0.37
Rumen	4.13	5.34	4.50	4.34	5.25	0.29[5]	0.006	0.03	0.02	0.66
Reticulum	0.60	0.63	0.58	0.56	0.68	0.03[5]	0.06	0.89	0.18	0.56
Omasum	1.71	1.79	1.77	1.60	2.27	0.12[5]	0.001	0.64	0.85	0.27
Abomasum	1.06	1.01	0.99	0.91	1.08	0.07[5]	0.89	0.45	0.80	0.41
Small intestine	3.54	3.63	3.91	3.73	3.68	0.18[5]	0.57	0.29	0.21	0.45
Large intestine	1.39	1.66	1.84	1.43	1.87	0.18[5]	0.06	0.10	0.41	0.08
Liver	3.25	3.56	3.71	3.66	3.86	0.14[5]	0.003	0.02	0.35	0.72
Pancreas	0.19	0.25	0.22	0.21	0.25	0.01[5]	0.003	0.01	0.03	0.64
Heart/lung	6.37	6.73	7.26	6.94	7.36	0.27[5]	0.01	0.06	0.11	0.35
Kidney	0.74	0.81	0.76	0.79	0.88	0.03[5]	0.001	0.17	0.17	0.43
Spleen	0.46	0.52	0.58	0.52	0.55	0.03[5]	0.02	0.01	0.07	0.07
Alimentary tract fat	4.64	4.75	5.16	5.93	5.40	0.31[6]	0.09	0.39	0.27	0.06
Omental fat	2.32	2.49	2.68	3.30	2.82	0.22[5]	0.09	0.30	0.49	0.03
Mesenteric fat	2.33	2.26	2.49	2.63	2.44	0.14[6]	0.58	0.82	0.18	0.40

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]Wet tissue weight.
[5]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[6]n = 8, except for LI-H_2O (n = 6), HI-H_2O (n = 6), and A-G (n = 7); SEM calculated using n = 6.

Table 4. Visceral organ and alimentary tract fat mass as a percentage of empty BW for steers fed at low intake (LI) or high intake (HI) and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM	HI-H_2O	R-, A-SH	A-SH	A-G
	% empty BW[4]									
Digestive tract	5.77	6.28	5.85	5.46	6.42	0.20[5]	0.02	0.22	0.09	0.14
Stomach complex	3.48	3.91	3.37	3.22	4.03	0.16[5]	0.01	0.39	0.008	0.46
Rumen	1.91	2.38	1.93	1.88	2.27	0.12[5]	0.03	0.10	0.005	0.77
Reticulum	0.28	0.28	0.25	0.24	0.29	0.01[5]	0.40	0.46	0.07	0.68
Omasum	0.80	0.80	0.76	0.70	0.99	0.05[5]	0.007	0.75	0.61	0.29
Abomasum	0.49	0.45	0.42	0.40	0.47	0.04[5]	0.58	0.19	0.53	0.53
Small intestine	1.65	1.62	1.69	1.62	1.59	0.08[5]	0.59	0.93	0.47	0.51
Large intestine	0.64	0.74	0.79	0.62	0.80	0.08[5]	0.12	0.16	0.59	0.08
Liver	1.51	1.59	1.60	1.59	1.67	0.05[5]	0.03	0.19	0.82	0.84
Pancreas	0.09	0.11	0.09	0.09	0.11	0.005[5]	0.02	0.06	0.004	0.75
Heart/lung	2.97	3.00	3.12	3.02	3.20	0.11[5]	0.12	0.50	0.32	0.42
Kidney	0.34	0.36	0.33	0.34	0.38	0.01[5]	0.05	0.96	0.05	0.39
Spleen	0.21	0.23	0.25	0.23	0.24	0.01[5]	0.11	0.07	0.19	0.12
Alimentary fat	2.15	2.11	2.22	2.57	2.34	0.13[6]	0.30	0.91	0.46	0.04
Omental fat	1.07	1.10	1.15	1.43	1.22	0.09[5]	0.20	0.57	0.68	0.02
Mesenteric fat	1.09	1.00	1.07	1.15	1.06	0.06[6]	0.71	0.49	0.33	0.35

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]Wet tissue weight as a percentage of empty BW.
[5]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[6]n = 8, except for LI-H_2O (n = 6), HI-H_2O (n = 6), and A-G (n = 7); SEM calculated using n = 6.

weights of total (i.e., muscle plus mucosal layers) and mucosal tissue for the duodenum and jejunum were unaffected by dietary MEI or treatments. Likewise, dietary intake did not affect ileal section weights. Total ($P = 0.03$) and mucosa ($P = 0.01$) weights of ileal sections were greater from steers receiving SH infusion, regardless of the site of carbohydrate infusion (R-SH vs. A-SH), than those from steers on the LI-H_2O treatment. Complexity of abomasally infused carbohydrate did alter ileal sections in that total ($P = 0.01$) and mucosa ($P = 0.02$) weights were greater for A-SH compared with A-G. Total colonic section weight was increased ($P = 0.02$) with SH infusion and tended ($P = 0.06$) to increase with dietary MEI but was unaffected by the site of SH infusion or form of abomasally infused carbohydrate. Colonic section mucosal weight was unresponsive to dietary MEI and carbohydrate infusion.

Whole ruminal tissue concentrations of DNA, RNA, and N were largely unaffected by treatment, with the exception of a trend for a greater ($P = 0.10$) DNA concentration for A-SH vs. A-G (Table 6). Consistent with slight directional changes in RNA and N concentrations, RNA:N tended to be greater for A-SH vs. R-SH ($P = 0.08$) and for A-SH vs. A-G ($P = 0.06$). Aside from a tendency for decreased ($P = 0.10$) whole jejunal tissue DNA concentration, dietary MEI did not alter DNA, RNA, and N concentrations of small intestinal whole tissue sections (i.e., duodenum, jejunum, and ileum). However, N:DNA for whole jejunal ($P = 0.04$) and ileal ($P = 0.02$) tissues increased with greater dietary MEI. No differences in small intestinal whole tissue concentrations of DNA, RNA, and N were observed between SH infusion and LI-H_2O. Site of SH infusion did not affect whole tissue duodenum and ileum concentrations of DNA, RNA, and N, with the exception of a trend ($P = 0.08$) for duodenal RNA concentration to be greater for R-SH compared with A-SH. However, whole ileal tissue DNA concentration increased ($P = 0.03$), and thus N:DNA decreased ($P = 0.03$) with A-SH vs. R-SH. Complexity of abomasal carbohydrate infusion did not affect whole jejunal tissue concentrations of DNA, RNA, and N. Similarly, complexity of abomasal carbohydrate infusion did not affect whole duodenal tissue concentrations of DNA, RNA, or N, but there was a trend ($P = 0.07$) for RNA:N to be greater for A-G compared with A-SH. Whole ileal tissue concentrations of RNA ($P = 0.04$) and RNA:N ($P = 0.03$) were greater for A-G vs. A-SH, whereas DNA and N concentrations were similar between abomasal treatments. Dietary MEI and infusion treatment did not affect whole colonic tissue concentrations of DNA, RNA, and N and N:DNA. However, SH infusion tended ($P = 0.09$) to decrease whole colonic tissue RNA:N compared with LI-H_2O. Likewise, whole colonic tissue RNA:N was less ($P = 0.05$) for A-SH vs. A-G.

Dietary MEI decreased ($P = 0.02$) hepatic epithelial concentration of DNA and increased N:DNA ($P = 0.0001$) but did not affect liver RNA and N concentra-

Table 5. Small intestinal length and total and mucosal mass of intestintal segments for steers fed at low intake (LI) or high intake (HI) and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM[4]	HI-H_2O	R-, A-SH	A-SH	A-G
Small intestine										
Length, m	29.8	32.3	29.4	28.4	31.9	1.0	0.13	0.39	0.03	0.42
Mass:length, g/m	118.2	113.0	133.2	131.6	115.6	5.0	0.70	0.41	0.002	0.80
Duodenum										
Total section, g	10.76	11.78	12.76	12.53	12.47	0.79	0.11	0.11	0.30	0.82
Mucosa, g	5.91	5.87	6.68	6.96	6.82	0.50	0.17	0.53	0.18	0.65
Jejunum										
Total section, g	12.83	11.54	13.17	13.24	12.49	0.87	0.77	0.64	0.13	0.95
Mucosa, g	7.24	6.28	7.14	7.76	7.14	0.69	0.91	0.51	0.30	0.47
Ileum										
Total section, g	15.95	17.93	19.79	16.04	16.57	1.09	0.67	0.03	0.16	0.01
Mucosa, g	7.66	9.31	10.77	8.44	8.69	0.75	0.30	0.01	0.11	0.02
Colon										
Total section, g	18.21	25.73	24.45	21.90	24.17	2.33	0.06	0.02	0.65	0.38
Mucosa, g	6.71	7.77	7.46	6.91	7.07	0.63	0.67	0.23	0.67	0.49

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.

tions or ruminal and intestinal mucosal concentrations of DNA, RNA, and N (Table 7). Rumen mucosal concentration of DNA was greater ($P = 0.05$) and N:DNA tended to be less ($P = 0.07$) for SH infusion compared with LI-H_2O, but concentrations of RNA and N were similar. In contrast, duodenum mucosal concentration of RNA ($P = 0.09$) and RNA:N ($P = 0.07$) and hepatic RNA ($P = 0.10$) tended to be greater for SH infusion vs. LI-H_2O, whereas concentrations of DNA and N were unchanged. Mucosal concentrations of DNA, RNA, and N in the remaining sections of the small intestine and colon were similar for SH infusion vs. LI-H_2O. Site of SH infusion did not alter concentrations of DNA and N in digestive tract mucosa and liver, with the exception of a trend ($P = 0.09$) for greater DNA concentration in jejunal mucosa for A-SH compared with R-SH. Similarly, mucosal concentrations of RNA were largely unaffected by the site of SH infusion, but ileal RNA:N ($P = 0.03$) and colonic RNA concentration ($P = 0.03$) and RNA:N ($P = 0.004$) were greater for R-SH vs. A-SH. With the exception of an increase ($P = 0.05$) in hepatic DNA concentration for A-SH vs. A-G, the form of carbohydrate did not affect mucosal concentrations of DNA, RNA, and N.

Hot carcass weights tended to be greater for steers receiving greater dietary MEI ($P = 0.09$) or SH infusion ($P = 0.08$) compared with steers on the LI-H_2O treatment (Table 8). Site and form of carbohydrate infused did not affect HCW. Longissimus muscle area was similar among treatments, with the exception of a greater ($P = 0.04$) LM area for A-G compared with A-SH. Increasing dietary MEI more than doubled ($P = 0.06$) subcutaneous fat thickness over the 5th rib but did not affect fat thickness over the 12th rib or concentration of intramuscular fat and KPH. Carbohydrate infusion, regardless of form or site of infusion, did not affect subcutaneous fat thickness. However, concentration of intramuscular fat was less ($P = 0.04$), and percentage of KPH tended to be less ($P = 0.06$), for SH infusion compared with LI-H_2O. Complexity of carbohydrate did not influence intramuscular fat concentration or percentage of KPH. However, intramuscular concentration of fat tended to be greater ($P = 0.10$) for A-SH vs. R-SH, whereas percentage of KPH fat was similar between the sites of SH infusion.

DISCUSSION

Experimental Model

Previous research has demonstrated a positive relationship between dietary MEI and increased maintenance energy costs associated with the visceral tissues (Ferrell et al., 1986; Burrin et al., 1992; Freetly et al., 1995). Specifically, digestive tract and liver tissue mass, as well as O_2 consumption, increase or decrease in accordance with increasing or deceasing amounts of dietary MEI (Burrin et al., 1989; Freetly et al., 1995; McLeod and Baldwin, 2000). Moreover, shifts in PDV O_2 consumption associated with MEI and dietary forage:concentrate ratios have been ascribed to differences in digestive tract organ weights rather than ruminal or intestinal cellular oxidative capacity (Baldwin and McLeod, 2000; McLeod and Baldwin, 2000), thus equating changes in PDV O_2 consumption to changes in organ mass. However, a direct cause-and-effect relationship

Table 6. Whole tissue DNA, RNA, and nitrogen content for steers fed at low intake (LI) or high (HI) intake and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs	LI-H_2O vs	R-SH vs	A-SH vs
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM	HI-H_2O	R-, A-SH	A-SH	A-G
Rumen										
DNA,[4] mg/g of tissue	7.25	7.93	8.67	6.30	7.35	1.11[5]	0.94	0.42	0.58	0.10
RNA, mg/g of tissue	6.33	7.08	8.85	6.57	7.69	1.09[5]	0.35	0.21	0.18	0.10
N, mg/g of tissue	114.3	120.9	118.8	111.8	118.9	8.7[5]	0.69	0.59	0.84	0.52
N:DNA	24.03	16.89	17.78	24.71	17.48	5.88[5]	0.40	0.34	0.90	0.35
RNA:N	0.053	0.058	0.076	0.057	0.064	0.008[5]	0.28	0.14	0.08	0.06
Duodenum										
DNA, mg/g of tissue	44.45	43.64	42.14	42.08	38.42	3.40[5]	0.19	0.70	0.71	0.99
RNA, mg/g of tissue	13.36	14.35	12.12	14.16	12.98	1.02[5]	0.78	0.92	0.08	0.12
N, mg/g of tissue	122.9	130.8	121.4	121.2	119.0	5.8[5]	0.61	0.64	0.18	0.98
N:DNA	2.85	3.10	3.04	2.91	3.25	0.19[5]	0.13	0.34	0.78	0.61
RNA:N	0.109	0.110	0.101	0.117	0.109	0.007[5]	0.96	0.68	0.30	0.07
Jejunum										
DNA, mg/g of tissue	46.05	43.34	48.42	50.81	35.80	4.62[5]	0.10	0.98	0.36	0.68
RNA, mg/g of tissue	11.50	12.13	12.06	13.11	10.49	1.07[5]	0.48	0.63	0.96	0.44
N, mg/g of tissue	123.2	130.2	129.9	127.9	118.1	6.4[5]	0.54	0.36	0.98	0.80
N:DNA	2.72	3.06	2.84	2.64	3.35	0.22[5]	0.04	0.38	0.41	0.47
RNA:N	0.094	0.093	0.093	0.103	0.088	0.007[5]	0.51	0.88	0.98	0.24
Ileum										
DNA, mg/g of tissue	68.80	57.09	73.51	74.86	57.54	6.80[6]	0.20	0.65	0.03	0.86
RNA, mg/g of tissue	13.31	13.38	14.69	17.52	13.28	1.14[6]	0.98	0.58	0.29	0.04
N, mg/g of tissue	125.3	130.2	132.7	133.8	137.2	5.5[6]	0.11	0.34	0.71	0.87
N:DNA	1.78	2.39	1.84	1.85	2.45	0.22[6]	0.02	0.18	0.03	0.97
RNA:N	0.110	0.103	0.111	0.131	0.096	0.008[6]	0.19	0.77	0.37	0.03
Colon										
DNA, mg/g of tissue	30.72	30.63	30.02	29.16	27.50	2.98[5]	0.42	0.91	0.86	0.82
RNA, mg/g of tissue	8.42	8.15	7.98	8.51	7.79	0.70[5]	0.49	0.67	0.84	0.54
N, mg/g of tissue	105.6	118.2	115.9	104.9	105.7	6.20[5]	0.99	0.12	0.76	0.17
N:DNA	3.44	4.00	4.03	3.72	4.11	0.31[5]	0.11	0.13	0.94	0.43
RNA:N	0.080	0.069	0.069	0.082	0.073	0.005[5]	0.30	0.09	0.98	0.05
Liver										
DNA, mg/g of tissue	16.68	16.95	16.93	14.92	14.01	0.79[5]	0.02	0.77	0.98	0.05
RNA, mg/g of tissue	9.13	10.51	9.81	9.28	9.68	0.52[5]	0.42	0.10	0.26	0.42
N, mg/g of tissue	112.5	117.1	110.8	106.3	117.7	4.7[5]	0.40	0.78	0.27	0.45
N:DNA	6.79	6.95	6.66	7.15	8.43	0.27[5]	0.0001	0.97	0.39	0.16
RNA:N	0.080	0.090	0.088	0.093	0.082	0.007[5]	0.85	0.31	0.84	0.56

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]Dry tissue basis.
[5]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[6]n = 8, except for LI-H_2O (n = 5) and A-G (n = 7); SEM calculated using n = 5.

between energy intake and visceral tissue mass, or O_2 consumption, is difficult to ascertain from data sets that have compared level of alimentation or dietary energy density because of confounding factors such as diet chemical composition and DMI. Case-in-point, Reynolds et al. (1991) reported that although PDV O_2 consumption increased with increasing dietary MEI, O_2 consumption was less in heifers fed a 75% concentrate vs. a 75% forage diet at equal MEI. This observed difference in O_2 use between diets reflects the cumulative impact of factors that affect visceral mass, potentially including site of energy substrate assimilation in the gastrointestinal tract. Among species, intestinal mass is influenced by inert bulk (Rompala et al., 1988), fermentable fiber (McCullogh et al., 1998), and a myriad of luminal and humoral factors (Johnson, 1987). Similarly, although less definitive, rumen mucosal mass has responded to VFA (Sakata and Tamate, 1978), especially during development (Lane et al., 2002), and total rumen mass and muscularity has been influenced by inert bulk (Hamada et al., 1976; Rompala et al., 1988). Assessing the quantitative impact of energy supply on visceral mass using a total infusion strategy does not appear to be a viable model because lambs maintained on total intragastric infusions of VFA mixtures and semipurified sources of protein and micronutrients exhibit intestinal atrophy (Orskov et al., 1979) or greatly reduced PDV O_2 use (Gross et al., 1990). Thus, for our experiment, a model was chosen that included feeding a basal diet, which allowed for growth and the mainte-

Table 7. Mucosal tissue DNA, RNA, and nitrogen content for steers fed at low intake (LI) or high (HI) intake and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM	HI-H_2O	R-, A-SH	A-SH	A-G
Rumen										
DNA,[4] mg/g of tissue	6.00	7.86	9.86	10.35	7.71	1.17[5]	0.27	0.05	0.16	0.74
RNA, mg/g of tissue	8.59	9.86	11.92	10.90	9.89	1.29[5]	0.45	0.14	0.19	0.53
N, mg/g of tissue	106.1	116.4	122.8	112.3	115.9	8.1[5]	0.37	0.17	0.51	0.31
N:DNA	19.33	15.73	14.31	11.76	16.61	1.99[5]	0.30	0.07	0.55	0.31
RNA:N	0.077	0.085	0.096	0.099	0.084	0.009[5]	0.58	0.24	0.33	0.82
Duodenum										
DNA, mg/g of tissue	54.95	59.21	64.57	59.20	56.40	5.03[5]	0.83	0.25	0.38	0.40
RNA, mg/g of tissue	17.99	20.65	20.26	18.78	18.36	1.22[5]	0.82	0.09	0.79	0.33
N, mg/g of tissue	128.2	128.4	128.4	125.9	127.2	3.30[5]	0.82	0.95	0.99	0.54
N:DNA	2.65	2.24	2.00	2.13	2.35	0.28[5]	0.42	0.12	0.48	0.71
RNA:N	0.140	0.161	0.158	0.149	0.143	0.009[5]	0.76	0.07	0.79	0.43
Jejunum										
DNA, mg/g of tissue	61.89	52.94	69.63	58.52	62.31	7.86[5]	0.97	0.95	0.08	0.26
RNA, mg/g of tissue	14.66	18.75	19.72	16.01	17.81	2.37[5]	0.32	0.11	0.73	0.22
N, mg/g of tissue	120.3	128.2	128.5	122.9	126.9	7.7[5]	0.51	0.37	0.98	0.56
N:DNA	1.99	2.27	1.94	2.13	2.20	0.17[5]	0.37	0.57	0.12	0.39
RNA:N	0.120	0.146	0.155	0.129	0.137	0.017[5]	0.49	0.15	0.66	0.24
Ileum										
DNA, mg/g of tissue	66.38	68.68	73.13	72.77	65.73	6.77[6]	0.94	0.56	0.54	0.96
RNA, mg/g of tissue	13.53	17.05	14.65	16.05	14.25	1.54[6]	0.71	0.20	0.16	0.42
N, mg/g of tissue	119.9	130.3	130.1	131.0	125.1	9.1[6]	0.67	0.34	0.98	0.94
N:DNA	1.76	1.92	1.82	1.81	1.91	0.10[6]	0.23	0.33	0.29	0.94
RNA:N	0.114	0.131	0.113	0.123	0.114	0.007[6]	0.98	0.34	0.03	0.23
Colon										
DNA, mg/g of tissue	55.92	61.45	58.51	56.68	54.24	6.42[5]	0.84	0.59	0.70	0.82
RNA, mg/g of tissue	13.94	18.40	14.24	14.77	15.55	1.51[5]	0.42	0.19	0.03	0.78
N, mg/g of tissue	113.9	124.1	120.1	112.4	112.2	8.7[5]	0.89	0.42	0.71	0.48
N:DNA	2.02	2.07	2.10	2.02	2.18	0.11[5]	0.29	0.64	0.84	0.56
RNA:N	0.123	0.149	0.119	0.130	0.136	0.008[5]	0.20	0.25	0.004	0.26

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of BW0.75·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]Dry tissue basis.
[5]n = 8 except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[6]n = 8 except for LI-H_2O (n = 5) and A-G (n = 7); SEM calculated using n = 5.

Table 8. Yield grade factors and intramuscular fat concentration for steers fed at low intake (LI) or high (HI) intake and infused ruminally (R) or abomasally (A) with hydrolyzed starch (SH) or glucose (G)[1,2]

							Contrast[3]			
	LI				HI		LI-H_2O vs.	LI-H_2O vs.	R-SH vs.	A-SH vs.
Item	H_2O	R-SH	A-SH	A-G	H_2O	SEM[4]	HI-H_2O	R-, A-SH	A-SH	A-Gt
HCW, kg	146.9	151.3	156.6	157.0	154.7	3.4	0.09	0.08	0.20	0.93
LM area, cm^2	47.5	48.3	47.3	53.2	50.4	2.2	0.32	0.90	0.68	0.04
Subcutaneous fat, mm										
5th rib	0.36	0.56	0.80	0.86	1.01	0.25	0.06	0.28	0.43	0.83
12th rib	0.43	0.36	0.59	0.53	0.59	0.19	0.54	0.85	0.31	0.78
LM i.m. fat,[5] mg/g of muscle	22.9	14.1	19.1	22.2	24.4	2.4	0.65	0.04	0.10	0.32
KPH, %	0.62	0.24	0.34	0.33	0.46	0.14	0.41	0.06	0.52	0.93

[1]Low and high intake represent 161 and 214 kcal of ME/(kg of $BW^{0.75}$·d), respectively.
[2]Isoenergetic infusion rates of SH [12.6 g/(kg of $BW^{0.75}$·d)] and G [14.4 g/(kg of $BW^{0.75}$·d)].
[3]Probability of larger *F*-statistic.
[4]n = 8, except for LI-H_2O (n = 6) and A-G (n = 7); SEM calculated using n = 6.
[5]Dry tissue basis.

nance of digestive tract integrity, supplemented with infused energy in the form of carbohydrate. This model was used to test 2 separate hypotheses. The first hypothesis is that digestive tract and liver mass are responsive to energy input with the extent of the response dependent upon site of carbohydrate infusion. The second hypothesis concerns our previous calorimetric observations that postruminal supply of SH results in a disproportionate amount of the energy available for tissue gain being deposited as adipose (McLeod et al., 2001). It is our hypothesis that a substantial portion of this increase in adipose tissue gain occurs in the visceral depots, and this is exacerbated by entry rate or simplicity of carbohydrate (i.e., monosaccharide vs. polysaccharide) delivered; this explains our rationale for inclusion of the postruminal glucose treatment.

Acute changes in digestive tract and liver mass and O_2 consumption in response to level of alimentation have been observed (Rompala and Hoagland, 1987; Burrin et al., 1990; Huntington et al., 1990). Notwithstanding the acute nature of these changes, a copious amount of time is required before complete adaptive responses are observed. Freetly et al. (1995) demonstrated that PDV and hepatic O_2 consumption reached steady-state 29 and 21 d, respectively, following dietary restriction. Conversely, using data obtained during a 42-d realimentation period of previously feed-restricted lambs, Freetly et al. (1995) reported that PDV and hepatic O_2 consumption reached asymptote at 38 to 40 d following realimentation to ad libitum intake. Accordingly, the 35-d treatment period in the current experiment was used so that changes in visceral organs would reflect completely adapted tissues and, based on our previous study (McLeod et al., 2001), would provide sufficient time to detect quantitative differences in indices of adipose accretion. Steers fed low (0.94 kg/d) or high (1.55 kg/d) dietary MEI gained BW over the 35-d treatment period, and as expected, carbohydrate infusion augmented BW gain (1.21 kg/d; average among infusion treatments) in steers fed LI. Although carbohydrate infusions were designed to be isoenergetic and rates were set to supply 20% of total MEI, the energy supplied by infusion of glucose slightly exceeded that supplied by SH; however, this did not alter total intake energy (Table 2) among carbohydrate infusion treatments. The amount of carbohydrate infused was based on a balance between maximal energy supply from SH and minimal quantities of glucose (as monomers or polymers) escaping the small intestine (Branco et al., 1999).

Visceral Organs

The positive relationship in our study between dietary alimentation and visceral organ mass is consistent with previous observations in cattle (Sainz and Bentley, 1997), sheep (Ferrell et al., 1986; Rompala et al., 1987; Burrin et al., 1990; McLeod and Baldwin, 2000), and pigs (Koong et al., 1983). The observed increase in digestive tract weight associated with dietary MEI was a function of greater individual organ weights with the exception of the abomasum and small intestine. Similarly, expression of organ weights as a function of EBW revealed an unsymmetrical increase in digestive tract with greater dietary MEI. This was largely due to increases in relative weights of organs that compose the stomach complex (i.e., rumen, reticulum, omasum, and abomasum), with the exception of the reticulum, and to a lesser extent (P = 0.12) the large intestine. The absolute and relative increases in digestive tract, stomach complex, and large intestinal mass in response to dietary MEI are consistent with our previous report in sheep (McLeod and Baldwin, 2000). However, in contrast to the current findings, McLeod and Baldwin (2000) reported that small intestinal mass in sheep was responsive to diet. Specifically, absolute and relative mass of the small intestine increased with greater dietary intake in lambs fed a 75% orchardgrass hay diet, but not in lambs fed a 75% concentrate diet. Considering that the basal diet (89.5% orchardgrass hay) in the current experiment was similar in constituents and physical form (i.e., pelleted) to the forage diet used by McLeod and Baldwin (2000), the reason for the unresponsiveness of small intestinal mass to dietary intake is not obvious. Aside from species differences, one explanation for the disparity between the current study and that of McLeod and Baldwin (2000) may be that the levels of dietary MEI used to test the effects of MEI on visceral organ mass were more extreme in the former (1-times vs. 2-times maintenance energy requirements) than in the current experiment (1.5 vs. 2.0 times maintenance energy requirements). Thus, in the former study, comparisons of small intestinal mass were made between sheep at zero and positive body tissue balance, whereas in the current experiment steers in both dietary intake treatment groups were in positive tissue balance.

Similar to that described for dietary MEI, increased digestive tract weight associated with SH infusion can be ascribed primarily to increased weight of the stomach complex (58%) with lesser contributions coming from the large (26%) and small (17%) intestine. However, in contrast to responses to dietary intake, these differences in organ weight were reconciled when expressed as a function of EBW. Alone, this suggests that SH infusion does not induce the same proliferative response in the digestive tract as does increased dietary intake. However, comparison between sites of SH infusion demonstrates that the effect of SH on digestive tract mass is highly dependent upon site of infusion. Although A-SH appeared to increase the thickness of the small intestine (Table 5; mass:length), a majority of which was accounted for by mucosa, the site of SH infusion did not affect small or large intestinal mass. On the contrary, expression of mass as a percentage of EBW demonstrates that R-SH disproportionally increased digestive tract and stomach complex mass relative to A-SH solely as a result of increased rumen mass. Taken together these data illustrate that digestive tract

mass is not simply a function of MEI, but rather is dependent upon site of energy substrate supply. The increase in rumen mass is presumably the result of luminal SH fermentation and subsequent VFA production. This presumption is consistent with studies that have shown VFA to stimulate rumen mucosa cell proliferation in adult (Galfi et al., 1991) and developing (Lane et al., 2002) ruminants. Another notable observation is the recognition that the greater partial efficiency of converting ME to tissue energy for intestinally vs. ruminally supplied SH (Harmon and McLeod, 2001) is not simply a function of fermentative and digestive energy losses, but also a reflection of the increased metabolic heat losses associated with maintaining greater rumen mass.

Interestingly, a posteriori comparison between R-SH and HI-H_2O reveals that rumen, reticulum, and abomasum mass, on an absolute weight basis or as a function of EBW, was similar ($P = 0.18$) between treatments, whereas omasum mass was greater ($P = 0.003$) for HI-H_2O than R-SH. Considering that dietary intake was less for R-SH compared with HI-H_2O, this suggests, at least within dietary intake parameters used in the current study, that the increase in rumen and reticulum mass observed with increased dietary intake is mainly a function of energy supply, whereas changes in omasum mass are related to dietary bulk or other factor(s). Although this is not definitively demonstrated, because it can be argued that ruminal energy digestibility may have been greater for R-SH, support is given to this contention by our previous observation that ME supplied by R-SH [196 kcal/(kg of $BW^{0.75}$·d)] did not exceed that of HI-H_2O [214 kcal/(kg of $BW^{0.75}$·d)] under the same experimental conditions (McLeod et al., 2001).

Generally, greater glucose supply and turnover results in a proportional increase in PDV glucose utilization (Seal and Reynolds, 1993). Indeed, using an experimental protocol similar to ours, Harmon et al. (2001) reported abomasal SH infusion increased PDV glucose utilization relative to ruminal SH infusion. Data from the current study verify that this is not the result of increased digestive tract mass. Controlled studies investigating the influence of intestinal supply of starch or glucose on intestinal mass are limited. However, continuous luminal supply of hyperosmolar glucose solutions (900 and 1,500 mos*M*) have increased small intestinal mucosa mass, with the greatest response occurring caudally, in rats maintained on total parenteral nutrition (Weser et al., 1985). It is difficult to make inferences about the contrasting results between the current study and that in rats because the experimental conditions are vastly dissimilar. Regardless, although neither A-SH nor A-G appeared to affect small intestinal mass, ileal total section and mucosa mass was greater for A-SH than A-G. Considering that large intestinal mass also tended to be greater for A-SH vs. A-G, these changes in mass likely represent differences in carbohydrate load presented to the caudal intestines (Branco et al., 1999).

Unlike R-SH, abomasal carbohydrate infusion did not influence reticulorumen mass. Previous efforts have demonstrated that abomasal, relative to ruminal, infusion of SH resulted in greater splanchnic energy output and peripheral glucose utilization by the PDV (Harmon et al., 2001). This implies that recticulorumen tissue proliferation is not responsive to arterial energy supply and that luminal VFA are the primary stimuli for proliferation. However, if recticulorumen tissue proliferation is sensitive to arterial oxidizable energy supply, the increase in splanchnic energy output (33.4 vs. 36.1 MJ/d), specifically arterial glucose concentration (4.87 vs. 4.59 m*M*) associated with abomasal SH infusion as observed by Harmon et al. (2001), may be insufficient to stimulate proliferation. Using isolated rumen epithelial cells, Baldwin and McLeod (2000) demonstrated that although these cells are capable of oxidizing glucose, the substrate concentration at which half Vmax oxidation rate is achieved for glucose (10.6 to 23.5 m*M*) was well above the arterial concentrations reported by Harmon et al. (2001).

A positive relationship between dietary MEI and rumen hyperplastic growth has been demonstrated (Burrin et al., 1992; McLeod and Baldwin, 2000; Baldwin et al., 2004). However, characterization of the cell population using N:DNA (Enesco and Leblond, 1962) and RNA:N (Burrin et al., 1992) as indices of cell size and protein synthetic capacity, respectively, has produced mixed results. Characterization of cellularity using whole rumen tissue (i.e., musculature and mucosa) from growing lambs (Burrin et al., 1992) or mucosa from lactating cows (Baldwin et al., 2004) showed dietary ME-induced mass changes were not accompanied by changes in cellular hypertrophy or protein synthetic capacity. In contrast, McLeod and Baldwin (2000) reported that rumen epithelial cells obtained from growing lambs tended to be smaller and had greater protein synthetic capacity compared with lambs fed to maintain BW. In the current experiment, increases in rumen mass, as a result of greater dietary MEI or R-SH, were not associated with reductions in whole or mucosal tissue concentrations of DNA, thus supporting previous findings of greater ruminal tissue cellularity. Moreover, the lack of change in N:DNA and RNA:N ratios in whole and mucosal tissue supports the contention that ME-induced increases in rumen mass are largely the result of hyperplasia without changes in cellular protein synthetic capacity.

Treatment-induced changes in cellularity of the caudal small intestine were apparent, suggesting that cell size or protein synthetic capacity, or both, were altered (Tables 6 and 7). However, these changes in cellularity resulting from dietary MEI and abomasal carbohydrate infusion were largely observed in whole jejunal and ileal tissue, but not mucosa, suggesting that these effects were relegated to the musculature. Given that jejunal section weights were unchanged by treatment and differences in ileal section weights are largely (>80%) explained by mucosal mass, the significance of

these changes in cellularity is not apparent. Similarly, inconsistencies in treatment effects on whole and mucosal colonic RNA concentrations and RNA:N were present and difficult to explain. Nevertheless, equable DNA concentrations indicate that the trophic effects of dietary MEI and SH supply on whole colonic tissue segments are the result of hyperplasia. It is unclear if this change in cellularity extends to the large intestine and thus characterizes the trophic effects of these treatments on the large intestine as a whole.

Although changes in heart and lung and spleen mass were proportionate to BW, those of the liver, pancreas, and kidney were not. The increase in liver mass resulting from greater dietary intake is consistent with our previous observation in sheep (McLeod and Baldwin, 2000). Liver mass has been shown to be responsive to dietary energy and protein (Wester et al., 1995), and thus overall metabolic workload. In this study, because dietary energy and protein are inextricably linked, the putative factor cannot be determined. However, considering that dietary energy and protein intake were identical for LI-H_2O and carbohydrate infusion treatments, combined with the fact that infusion treatments provided an intermediate amount of total intake energy relative to low and high dietary intake controls, the intermediate increase in liver mass associated with carbohydrate infusion, perhaps with the exception of R-SH (discussed directly below), corroborate the linkage between energy supply and liver mass. The increase in liver mass associated with dietary MEI appeared to be a function of cellular hypertrophy, whereas that which occurred in response to SH infusion was due to cellular hyperplasia. Similar to liver, observed increases in pancreas and kidney mass with greater MEI could potentially be a function of protein or energy supply. The greater mass for both organs with R-SH compared with A-SH argues that protein supply may be the dominant factor considering that microbial protein flow would be enhanced by R-SH. This is supported by studies demonstrating sensitivity of pancreas (Swanson et al., 2003) and kidney mass (Wester et al., 1995) to protein supply.

Adiposity

Previously we have demonstrated that R- and A-SH infusion increased retained tissue energy above that observed with the same basal diet employed in the current study, with the greatest retention occurring with A-SH (McLeod et al., 2001). Partitioning of the increased retained tissue energy, using C-N balance techniques, revealed retained energy deposited as protein and lipid was 30 and 70% for R-SH and 16 and 84% for A-SH. Because the amount of tissue energy retained as protein was similar for SH treatments, the observed differences in the proportions of retained tissue energy as protein and lipid were directly a function of greater retained tissue energy with A-SH. As such, the observed increase in retained tissue energy for A-SH compared with R-SH was accounted for solely as adipose tissue. Consistent with this observation, Owens et al. (1986), citing unpublished data, reported that abomasal glucose infusion resulted in greater retained tissue energy, 87% of which was retained as lipid, compared with lambs fed equal amounts of glucose. In the current study, indices of adiposity (i.e., alimentary fat mass, subcutaneous fat thickness, KPH, and intramuscular fat) were largely unaffected by SH infusion, the exception being a decrease in intramuscular fat with R-SH compared with all other treatments. Nevertheless, the numerical patterns among indices are in general agreement with our previous findings of greater adiposity with A-SH compared with R-SH (McLeod et al., 2001). Because only alimentary adipose tissue mass was quantified, and not whole body lipid, a definitive agreement between studies is precluded.

The observed changes in absolute and relative amounts of alimentary fat mass, as well as numerical trends in carcass indices, indicate that abomasally supplied glucose stimulated lipid accretion to a greater extent than R- and A-SH. This is particularly evident in the comparison of A-SH vs. A-G. Two putative explanations for the increase in lipid accretion with A-G are an increase in glucose absorption and a greater supply of energy available for tissue accretion. Although circulating blood glucose concentrations were not measured in the current experiment, concentrations may have been augmented by A-G to a greater extent than SH infusion. Net splanchnic output of glucose (201 vs. 95 mmol/h) is greatly enhanced in steers when SH is digested in the small intestine as opposed to the rumen (Harmon et al., 2001). Additionally, intestinal disappearance data demonstrate that as much as 12% of SH flows past the ileum (Branco et al., 1999), and thus, availability of glucose for absorption, and ultimately, circulating glucose concentrations, may have been greater for A-G than for A-SH. Given the potential differences in circulating glucose concentrations, it is possible that insulin secretion may have been altered. Insulin has been shown to have a permissive effect on adipose accretion in that it stimulates the uptake of glucose and acetate by muscle and adipose tissue. However, the major role in insulin on adipose accretion in ruminants is mediated via antilipolytic actions rather than stimulation of fatty acid synthesis (Brockman, 1986). In the current study, based on samples collected at the end of the 35-d infusion period, insulin concentrations were unchanged by treatment (Baldwin et al., 2007). This may be reflective of the fact that VFA and glucose are secretagogues for insulin (Brockman, 1986). The lack of change in circulating blood concentrations of insulin does not negate the possibility that glucose may have had a direct effect on lipid accretion. Glucose has been shown to stimulate expression of lipogenic enzyme mRNA (fatty acid synthetase, EC 2.3.1.85; acyl-CoA carboxylase, EC 6.4.1.2) in rat adipose tissue independent of insulin via intracellular glucose-6-phosphate concentrations (Girard et al., 1997). Given the differ-

ences in adiposity, combined with the aforementioned 2-fold potential difference in splanchnic glucose output between R-SH vs. A-SH, it is questionable if the potential difference in intestinal glucose uptake (12%) between A-G and A-SH would sufficiently account for observed differences in adiposity. An alternative explanation is that A-G may have supplied a greater amount of energy for tissue accretion. Likewise, the same could be stated for A-SH compared with R-SH. Based on previously determined values of ME and partial efficiencies of converting ME to tissue energy for the basal diet and for ruminally and abomasally infused SH (McLeod et al., 2001), our A-SH treatment supplied 174 kcal/kg of $BW^{0.75}$ more tissue energy than R-SH. This extra tissue energy supply represents 1.28 kg of adipose accretion (assuming 9.5 kcal/g of fat) of which 33% was accounted for by observed differences in alimentary adipose depots. Comparable values for A-G have not been determined previously; however, adjusting the partial efficiency of A-SH for heat loss due to glucosidic bond cleavage (4.3 kcal/mol of starch; Baldwin, 1968) and accounting for the depression in dietary energy intake suggests no difference in tissue energy between A-SH and A-G and, thus, supports an energy independent mechanism for increased adiposity. Adjusting ME for differences in DE between A-G and A-SH (99 vs. 88% of infused energy, Branco et al., 1999) and a partial efficiency of converting ME to tissue energy (0.685) adjusted for small intestinal glucose disappearance (Harmon and McLeod, 2001) and glucosidic bond cleavage, yields an estimated increase of 331 kcal/kg of $BW^{0.75}$ available for adipose accretion or 2.4 kg of adipose of which 35% is accounted for by alimentary adipose differences. Although carcass fat indices are consistent with a general increase in whole body lipid accretion for A-SH compared with R-SH and A-G compared with A-SH, it is equivocal whether differences in lipid accretion are a function of tissue energy supply or substrate driven because whole body lipid was not quantitatively measured.

Although carcass fat was not quantitatively determined, comparing the relative increases in carcass fat indices with that of alimentary adipose tissue mass, specifically omental fat mass, observed with A-G is suggestive of a differential response to glucose supply among the different depots. It is unlikely that these differences are reflective of lipolysis rate, because absolute or fractional rates of lipolysis between these depots have been shown to be similar in ruminants (Prior, 1978) and elevated in the alimentary depot of humans (Turner et al., 2003). It has been demonstrated that although the contribution of glucose carbon to fatty acid synthesis in adipose in vitro is low relative to acetate, the contribution is greater for intramuscular than subcutaneous adipose (Smith and Crouse, 1984). Incubation of adipose tissues obtained from steers in the current A-G treatment group extends this observation in that although in vitro rates of acetate utilization for fatty acid synthesis were greater than that observed for glucose, in vitro rates of glucose utilization were greater for omental compared with subcutaneous adipose tissue (Baldwin et al., 2007). This is consistent with measures of glucose uptake in humans, as determined by positron emission tomography with labeled [^{18}F]fluoro-deoxy-glucose, which show that glucose uptake by visceral adipose tissue is greater than subcutaneous adipose (Virtanen et al., 2002). Additionally, A-G induced increases in the transcription of genes encoding for lipogenic regulatory nuclear proteins carbohydrate response element binding protein, sterol regulatory element-binding protein 1, and spot 14, as well as their established targets, fatty acid synthetase and acyl-CoA carboxylase (Baldwin et al., 2006). Relative to LI-H_2O, increases in transcript numbers for these genes ranged from 1.1- to 2.5-fold for subcutaneous and 3.5- to 9.7-fold for omental adipose tissue. Taken in concert, our in vivo, in vitro, and gene transcript observations are supportive of the concept that the omental adipose tissue is a preferred storage depot when glucose energy supply is sufficiently elevated in growing steers.

In conclusion, previous research has demonstrated that visceral organ mass is responsive to level of alimentation and dietary chemical composition. This research extends these findings by demonstrating that increasing ruminal energy supply results in a disproportionate increase in rumen mass. In contrast, increasing small intestinal energy supply does not elicit changes in digestive tract organ mass. However, small intestinal energy supply, especially in the form of glucose, results in greater adipose accretion compared with energy supplied ruminally, and the omental, as opposed to subcutaneous or intramuscular, adipose appears to be the preferred site for storage. Prediction models should be reparameterized to account for heat losses associated with maintenance of increased rumen tissue and differences in composition of accreted tissue observed with alterations in site of carbohydrate digestion.

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